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#60	Search **baculovirus and HBV and compound** Limits: **Publication Date to 2000/06/09**	14:56:55	0
#57	Search **AD cell line and HBV** Limits: **Publication Date to 2000/06/09**	14:53:12	20
#56	Search **AD cell line** Limits: **Publication Date to 2000/06/09**	14:53:01	5385
#53	Search **AD and HBV and compound** Field: **All Fields,** Limits: **Publication Date to 2000/06/09**	14:50:34	3
#52	Search **AD and HBV and compound**	14:50:21	4
#50	Search **Doong S 1991**	14:25:43	1
#30	Search **Boyd 1987 and HBV**	12:03:38	0
#49	Search **Kruger 1994 and HBV**	12:03:11	2
#48	Search **main 1996 and HBV**	12:02:33	32
#47	Search **Severini 1995 and HBV**	12:02:20	1
#46	Search **Dienstag 1995 and HBV**	12:02:03	2
#45	Search **Bock 1997 and HBV**	12:01:43	1
#44	Search **Nakbayashi 1982 and HBV**	12:00:43	328
#43	Search **Trautwein 1993 and HBV**	12:00:20	1
#42	Search **Bruss 1991 and HBV**	12:00:00	1
#41	Search **Briss 1991 and HBV**	11:59:52	0
#40	Search **Okamoto 1992 and HBV**	11:59:18	7
#39	Search **McMahon 1992 and HBV**	11:59:03	0
#38	Search **Norder 1993 and HBV**	11:58:38	4
#37	Search **Poch 1989 and HBV**	11:58:22	0
#36	Search **Poch 1989**	11:58:12	11
#35	Search **Bartholomeusz 1997**	11:57:50	3
#34	Search **Tillmann 1999 and HBV**	11:57:13	4
#33	Search **Tillmann 1999**	11:57:07	34
#32	Search **Hodge V 1993**	11:56:50	0

#31 Search **Hodge V 1993 and HBV**	11:56:45	0
#29 Search **Vere D 1993 and HBV**	11:56:16	0
#28 Search **Hodge 1993 and HBV**	11:56:08	0
#27 Search **Vere Hodge 1993 and HBV**	11:56:01	0
#26 Search **Summers 1982 and HBV**	11:55:17	7
#25 Search **Summers 1982**	11:55:08	74
#23 Search **Torresi J and HBV** Limits: **Publication Date to 2000/06/09**	10:50:27	1
#22 Search **Alister S and HBV** Limits: **Publication Date to 2000/06/09**	10:50:08	0
#20 Search **Trautwein C and HBV** Field: **All Fields,** Limits: **Publication Date to 2000/06/09**	10:45:33	14
#19 Search **Trautwein C and HBV**	10:45:16	28
#18 Search **Trautwein C and HBV and inhibit**	10:45:05	2
#16 Search **Manns M and HBV treatment**	10:44:32	32
#17 Search **Manns M and HBV and inhibit**	10:43:59	2
#15 Search **Manns M and HBV**	10:43:29	64
#14 Search **Tillmann H and HBV**	10:43:00	30
#12 Search **Bock T and HBV**	10:40:43	7
#11 Search **Bock T**	10:40:36	72
#6 Search **anti-HBV and cell line and screening**	10:06:07	4
#5 Search **anti-HBV and cell line**	10:05:59	59
#2 Search **anti-HBV and detect**	09:53:17	4
#1 Search **anti-HBV drug**	09:52:34	147

Clear History

```
=> HBV
L1        19429 HBV

=> screening (w) assay
L2         4580 SCREENING (W) ASSAY

=> L1 and L2
L3           24 L1 AND L2

=> inhibit (l) L1
L4          637 INHIBIT (L) L1

=> assay
L5       808989 ASSAY

=> L4 and L5
L6          125 L4 AND L5

=> cell (l) line
L7       692543 CELL (L) LINE

=> L1 and L7
L8         1693 L1 AND L7

=> L8 and L6
L9           47 L8 AND L6

=> vector or plasmid
L10      503456 VECTOR OR PLASMID

=> L10 and L1
L11        2220 L10 AND L1

=> L5 and L11
L12         302 L5 AND L11

=> L12 and L9
L13          10 L12 AND L9

=> D L10 IBIB ABS 1-10
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=> "drug screening"
L14       27763 "DRUG SCREENING"

=> L1 and L14
L15          57 L1 AND L14

=> inibit and L15
L16           0 INIBIT AND L15

=> inhibit and L15
L17          13 INHIBIT AND L15

=> D L17 IBIB ABS 1-13

L17  ANSWER 1 OF 13  CAP
```

L13 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:402599 BIOSIS
DOCUMENT NUMBER: PREV199900402599
TITLE: Specific inhibition of hepatitis B virus replication by sense RNA.
AUTHOR(S): Putlitz, Jasper Zu; Wands, Jack R. [Reprint author]
CORPORATE SOURCE: Liver Research Center, Rhode Island Hospital, Brown University School of Medicine, 55 Claverick Street, Providence, RI, 02903, USA
SOURCE: Antisense and Nucleic Acid Drug Development, (June, 1999) Vol. 9, No. 3, pp. 241-252. print.
ISSN: 1087-2906.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Oct 1999
Last Updated on STN: 8 Oct 1999

AB We describe effects of sense RNA molecules on hepatitis B virus (**HBV**) replication and antigen synthesis in transiently transfected cells. When certain subgenomic fragments of **HBV** were expressed as sense RNA together with a replication-competent genome of **HBV** , they inhibited **HBV** replication by up to 75% and HBsAg secretion by up to 60%. The corresponding antisense sequences had a 50% inhibitory effect in one case and no effect in another case. The sense RNA species did not **inhibit** duck hepatitis B virus (DHBV) replication, suggesting specific inhibitory effects. **HBV** transcript levels were unaltered in the presence of sense RNA species, consistent with an inhibitory effect mediated at the posttranscriptional level. The inhibition of **HBV** replication by overexpression of sense RNA derived from the viral genome represents an example of sense cosuppression of an animal virus.

L13 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:330563 BIOSIS
DOCUMENT NUMBER: PREV200300330563
TITLE: Construction of hepatocellular specific preS2 antisense RNA expression **vector** and study of its inhibitory effect.
AUTHOR(S): Ma Chun-hong; Sun Wen-sheng [Reprint Author]; Zhang Li-ning; Cao Ying-Lin; Wang Zhen-guang; Liu Su-xia; Wang Xiao-Yan; Zhu Fa-liang
CORPORATE SOURCE: Medical School, Institute of Immunology, Shandong University, Ji-nan, 250012, China
sunwenshengsws@yahoo.com
SOURCE: Zhonghua Weishengwuxue He Mianyixue Zazhi, (April 2003) Vol. 23, No. 4, pp. 279-282. print.
CODEN: ZWMZDP. ISSN: 0254-5101.
DOCUMENT TYPE: Article
LANGUAGE: Chinese
ENTRY DATE: Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003

AB Objective: To obtain recombinant preS2 antisense RNA expression **vector** targeted to hepatocellular carcinoma. Methods: preS2 gene (3203-340) was obtained by PCR and then cloned into EB virus **vector** pEBAF containing human alpha-fetoprotein promoter and enhancer. After transfection by lipofectin, RNA dot blot, ELISA and real-time qualitative PCR were used to **assay** the expression preS2 mRNA, **HBV** antigens and **HBV** DNA respectively. Results: The recombinant pEBAF-as-preS2 which can be expressed specifically in hepatocellular carcinoma **cells** was successfully obtained. Both **HBV** DNA and **HBV** antigens of hepatocellular **cell line** HepG2.2.15 were inhibited by transfection by pEBAF-as-preS2. The inhibition rate of HBsAg, HBeAg and **HBV** DNA were 33.4%, 41.5% and 86% respectively. Conclusion: pEBAF-as-preS2 expression **vector** may lead to targeted-expression of preS2 antisense RNA on hepatoma **cells** and **inhibit** **HBV** multiplication.

L13 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:473074 CAPLUS
DOCUMENT NUMBER: 129:271193
TITLE: Inhibition of hepatitis B virus by retroviral **vectors** expressing antisense pre-C/C gene fragment
AUTHOR(S): Ji, Wei; Wang, Qinhuan; Si, Chongwen; Zhang, Guoqing
CORPORATE SOURCE: The Fourth Department of Infectious Diseases, The 302nd Hospital of the PLA, Beijing, 100039, Peop. Rep. China
SOURCE: Zhongguo Bingduxue (1997), 12(3), 214-218
CODEN: ZBINER; ISSN: 1003-5125
PUBLISHER: Kexue Chubanshe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB Retroviral **vector** expressing antisense RNA complementary to hepatitis B virus (**HBV**) DNA pre-C/C region (ANTI-C) was used to transduce the human hepatoblastoma **cell line** 2.2.15, which was transfected with **HBV** DNA and can express **HBV** markers. The inhibitory effects of antisense gene mediated by retroviral **vector** ANTI-C on the expression of **HBV** antigens appeared as early as on the 3rd day after transduction, reached peak level on the 5th day, and persisted at least for 11 days. The inhibitory rates of HBsAg and HBeAg expression in 2.2.15 **cells** by ANTI-C were 27.0% and 59.5% on the 5th day after transduction. **HBV** DNA in the supernatant of 2.2.15 **cells** transduced with ANTI-C also reduced on the 5th day after transduction as detected by DNA dot blot **assays**. The viability of transduced 2.2.15 **cells** was not affected. Antisense retroviral **vectors** containing antisense gene can **inhibit HBV** and may be potentially useful for anti-**HBV** gene therapy.

L13 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2004:134204 BIOSIS
DOCUMENT NUMBER: PREV200400132270
TITLE: LY582563 (MCC-478), a 2-aminopurine nucleotide analogue, **inhibits** packaging and replication of **HBV** genome in vitro.
AUTHOR(S): Shaw, Tim [Reprint Author]; Colledge, Danni [Reprint Author]; Sozzi, Vitina [Reprint Author]; Locarnini, Stephen A. [Reprint Author]
CORPORATE SOURCE: Victorian Infectious Diseases Reference Laboratory, North Melbourne, VIC, Australia
SOURCE: Hepatology, (October 2003) Vol. 38, No. 4 Suppl. 1, pp. 716A. print.
Meeting Info.: 54th Annual Meeting of the American Association for the Study of Liver Diseases. Boston, MA, USA. October 24-28, 2003. American Association for the Study of Liver Diseases.
ISSN: 0270-9139 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Mar 2004
Last Updated on STN: 10 Mar 2004

AB Background: LY582563, a new synthetic nucleotide analogue developed jointly by Mitsubishi Pharmaceutical Company and Eli Lilly, is a potent inhibitor of replication of both wild type (wt) and lamivudine (LMV) resistant **HBV** strains. Prior experiments indicate that its mechanism(s) of action differs from those of LMV and adefovir. Aims: To investigate the mechanisms by which LY582563 **inhibits** **HBV** replication, in vitro. Methods: Compound LY58263 was provided by Eli Lilly. The following viral parameters were investigated: (1) Endogenous **HBV** polymerase activity was measured in **cell**

free **assays** using isolated, partly purified virus particles as the enzyme source. (2) Biosynthesis of viral antigens and replicative intermediates including **HBV** ccc DNA were studied in HepG2 **cells** transduced with wt **HBV** using a recombinant baculovirus **vector**. (3) Three derivatives of the Huh-7 **cell line**, in which the expression of **HBV** core protein is controlled by a tetracycline responsive element (pTRE) were used to study packaging in trans. These were: pTRE (control), PC47 (precore producing; no rescue control), C4B (core producing; will rescue). (4) AD-38 HepG2 **cells**, in which expression of an integrated **HBV** genome occurs in response to tetracycline withdrawal, were used to compare the antiviral effects of LY582563 and interferon-alpha (IFN). Production of **HBV** RNA, intra- and extra- cellular DNA and nuclear ccc DNA was monitored by Northern blotting, quantitative RT-PCR (QPCR), and Southern blotting respectively. Viral antigens were detected and quantified by SDS-PAGE and immunoblotting. Southern blotting, QPCR, immunoblotting and electron microscopy (EM) were used to study capsid formation and encapsidation of pregenomic RNA (pgRNA). Results: Phosphorylated metabolites of LY582563 did not **inhibit** endogenous **HBV** DNA polymerase activity. LY582563 had no significant effect on generation of **HBV** cccDNA, RNA, or the expression of viral antigens (HBe, HBs or HBc) in the recombinant baculovirus system. Inhibition of packaging of the pgRNA in cis, but not in trans, was observed in both baculovirus and pTRE based **assays** . EM examination revealed that the effect on packaging was not due to inhibition of core dimerisation or production of nucleocapsids. Furthermore, the antiviral effect of LY582563 in AD38 HepG2 **cells** did not appear to be mediated by an IFN-like mechanism. Conclusions: LY582563 interfered with the packaging of **HBV** pgRNA into nucleocapsids. Since correctly assembled nucleocapsids are a prerequisite for efficient **HBV** replication, interference with the packaging of **HBV** pgRNA by LY582563 may be the primary mechanism by which it **inhibits HBV** replication. Further studies are required to determine whether LY582563 specifically or differentially **inhibits** priming of reverse transcription or RNase H activity. Based on this novel mechanism of action and its activity against wt and LMV-resistant **HBV**, further clinical studies of LY582563 appear warranted.

L13 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:185369 CAPLUS
DOCUMENT NUMBER: 131:17201
TITLE: Hepatitis B virus X protein inhibits nucleotide excision repair
AUTHOR(S): Jia, Libin; Wang, Xin Wei; Harris, Curtis C.
CORPORATE SOURCE: Laboratory of Human Carcinogenesis, Division of Basic Sciences, National Institutes of Health, Bethesda, MD, USA
SOURCE: International Journal of Cancer (1999), 80(6), 875-879
CODEN: IJCNAW; ISSN: 0020-7136
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Human hepatitis B virus (**HBV**) is a major risk factor of human hepatocellular carcinoma. Both in vivo and in vitro studies have shown that **HBV** X protein (HBx) can bind to the p53 tumor-suppressor protein and interfere with the role that p53 plays in the cellular response to DNA damage. Our previous work has shown that HBx protein **inhibits** p53 sequence-specific transcriptional activation, p53-mediated apoptosis and p53 binding to the TFIIH transcription-nucleotide excision repair (NER) factors, including XPB and XPD. To investigate whether HBx interferes with the NER pathway, we utilized **cell**-proliferation and colony-formation **assays** to determine if **cells** expressing HBx are more sensitive to UVC-induced DNA damage. NER was also measured by a **plasmid** host **cell** re-activation **assay** using a **vector** containing a luciferase reporter gene. UV-irradiated **plasmids** were transfected into a human RKO colon carcinoma **cell line** that contains wild-type (wt) p53 as well as its derivs., either mutant p53-143ala (RKO-143ala) or human papillomavirus E6 (RKO-E6, a wt p53 protein that is rapidly degraded and non-functional). We found that **cells** expressing HBx are more sensitive to UVC-induced killing. Moreover, expression of HBx resulted in a reduction of NER efficiency in RKO **cells** to 52±2% (compared with control), RKO-143ala **cells** to 46±3% and RKO-E6 **cells** to 60±3%. Similar results were also obtained with a HepG2 hepatoblastoma **cell line** carrying wt p53. In addition, we found that HBx bound directly to either XPB or XPD DNA helicase in vitro. Thus, our data indicate that HBx may interfere with the NER pathway through both p53-dependent and p53-independent mechanisms. Because HBx binds to TFIIH-associated proteins, we propose that HBx may interfere with the NER pathway also through binding to and altering the activities of helicases necessary for NER and, thereby, increase the mutation rate induced by chemical carcinogens, such as aflatoxin B1, during human liver carcinogenesis.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:703627 CAPLUS
DOCUMENT NUMBER: 140:179972
TITLE: Inhibition of **HBV** by ribozyme in adenovirus expression system
AUTHOR(S): Li, Jinge; Zhang, Rong; Zhou, Yongxing; Lian, Jianqi; Wang, Jiuping
CORPORATE SOURCE: Tangdu Hospital, Fourth Military Medical University, Xi'an, 710032, Peop. Rep. China
SOURCE: Jiefangjun Yixue Zazhi (2003), 28(3), 243-245
CODEN: CFCHBN; ISSN: 0577-7402
PUBLISHER: Jenminjun Chubanshe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The inhibition of **HBV** expression by multi-target ribozyme-expressing adenovirus **vector** was studied in vivo. The ribozyme and muta-ribozyme were cloned into adenovirus **vector** pAd-CMV. By using the adenovirus expression system, the two **plasmids** were co-transfected into 293 **cell line** with adenovirus gene recombinant **plasmids** pJM17. The recombinant adenovirus were detected by plaque **assay**. Then the 2.2.15 **cell** were infected with recombinant adenovirus. Using ELISA, dot-bolt hybridization and image anal. system, the inhibition of **HBV** expression was detected. The ribozyme and muta-ribozyme were cloned into the adenovirus **vector** pAd-CMV. The recombinant adenovirus could infect 2.2.15 **cells** and express the ribozyme. The ribozyme could decrease the expression of HBeAg by 87.1%. These results indicate that ribozyme can **inhibit HBV** expression in the **cell**, which represents a potential gene therapy for **HBV** infection.

L17 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:592171 CAPLUS
DOCUMENT NUMBER: 130:10292
TITLE: Study on screening anti-**HBV** agents from antisense oligodeoxynucleotide and their toxicity
AUTHOR(S): Han, Jinxiang; Wang, Yulin; Zhang, Cui
CORPORATE SOURCE: Shandong Medicinal Biotechnology Centre, Jinan, 250062, Peop. Rep. China
SOURCE: Zhongguo Shenghua Yaowu Zazhi (1998), 19(3), 131-135
CODEN: ZSYZFP; ISSN: 1005-1678
PUBLISHER: Zhongguo Shenghua Yaowu Zazhi Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB To screen a new anti-**HBV** agent, according to sequence of **HBV** genome, 5 complementary sites were selected and the antisense phosphorothioate oligodeoxynucleotides (s-asODN) were designed and synthesized, they are S gene initiator, SP II cap site, Pre-C gene initiator, C gene initiator and gene ε initiator. The complementarity to different **HBV** genome were assayed for their anti-**HBV** activity in HepG22215 cell line with ELISA method, and the toxicity of s-asODN also was studied with MTT colorimetric assay and electron microscope. The result shows that s-asODN can **inhibit** the expression of HBsAg and HBeAg, and the inhibition shows sequence-specific, dose-related, antinuclease and combination-cooperative, and harmless to host characteristic in the viability, the shape and ultrastructure of cells. The study had laid the foundation of exploring new anti-**HBV** agents.

ANSWER 10 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:227816 CAPLUS
DOCUMENT NUMBER: 132:261362
TITLE: A quick in vitro assay for viral DNA polymerase activity of hepatitis B virus and its use in antiviral **drug screening**
INVENTOR(S): Oon, Chong Jin; Chen, Wei Ning; Lim, Gek Keow; Leong, Ai Lin
PATENT ASSIGNEE(S): Government of the Republic of Singapore, Singapore
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000018958	A2	20000406	WO 1998-SG77	19980929
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9893726	A1	20000417	AU 1998-93726	19980929
CA 2344282	AA	20000406	CA 1999-2344282	19990313
WO 2000018968	A1	20000406	WO 1999-SG17	19990313
WO 2000018968	C1	20021107		
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AU 9929694	A1	20000417	AU 1999-29694	19990313
TR 200100899	T2	20010723	TR 2001-200100899	19990313
EP 1117843	A1	20010725	EP 1999-910940	19990313
R: AT, BE, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE, PT, IE, FI				
BR 9914099	A	20010731	BR 1999-14099	19990313
JP 2002525130	T2	20020813	JP 2000-572415	19990313
NZ 510704	A	20021025	NZ 1999-510704	19990313
RU 2202620	C2	20030420	RU 2001-108496	19990313
NO 2001001510	A	20010528	NO 2001-1510	20010323
HR 2001000230	A1	20020630	HR 2001-230	20010327
US 6593082	B1	20030715	US 2001-674465	20010618
US 2003082529	A1	20030501	US 2002-231405	20020828

PRIORITY APPLN. INFO.:

WO 1998-SG77	A	19980929
WO 1999-SG17	W	19990313
US 2001-674465	A3	20010618

AB The hepatitis B virus (**HBV**) DNA polymerase for the assay is obtained from coupled in vitro transcription/translation of PCR products of serum derived **HBV** genomic DNA using primers containing SP6 RNA polymerase promoter sequence. The **HBV** DNA polymerase activity is measured by filtration assay for the priming activity in the presence of a radio-labeled nucleotide. The assay is tested with two **HBV** escaping mutants containing mutation in 'a' epitope of HBsAg (not protected by the vaccines, 's' 126 or 's' 145) and also used for screening for antiviral drugs that **inhibit HBV** DNA polymerase.

L17 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:368224 CAPLUS
DOCUMENT NUMBER: 136:83781
TITLE: Hepatitis B virus transgenic mice for model of anti-hepatitis B virus drug study
AUTHOR(S): Xiong, Yili; Jia, Yanzheng; Wang, Hongmin; Liu, Guangze; Ren, Hong; Zhou, Zhi; Zhang, Dingfeng
CORPORATE SOURCE: Transgenic Engineering Lab, infectious Disease Center of PLA, 458th Hospital of PLA, Canton, 510602, Peop. Rep. China
SOURCE: Zhonghua Ganzangbing Zazhi (2001), 9(1), 19-21
CODEN: ZGZZFE; ISSN: 1007-3418
PUBLISHER: Chongqing Yike Daxue, Dier Linchuang Xueyuan
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB The hepatitis B virus (**HBV**) transgenic mice models was established for studying if the model can be used for the evaluation of anti-**HBV** drugs. **HBV** transgenic mice models were produced by microinjection to analyze the integration, expression of **HBV** in the transgenic mice by nested PCR, southern blot, immunohistochem., and ELISA. Sixty mice whose **HBV** DNA, HBsAg were pos. were divided into 6 groups randomly, in which 3 groups were given drugs: lamivudine administered by perfusion of stomach tube (100 mg.kg- 1. day-1 for 21 days); thymosin administered by abdomen injection (3 mg.day-1 for 90 days); and DNA vaccine of 100μ g by muscle injection. The other 3 groups were neg. control. Lamivudine, thymosin and DNA vaccine made **HBV** DNA become neg. in the serum of **HBV** transgenic mice. The neg. ratio was highest in lamivudine treatment group. **HBV** DNA became pos. again when lamivudine terminated. Lamivudine, thymosin, and DNA vaccine can **inhibit** **HBV** replication. Transgenic mice might be used as the model for anti-**HBV** **drug screening** and eval

L17 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:798297 CAPLUS
DOCUMENT NUMBER: 135:352761
TITLE: Synthetic peptides that bind to the hepatitis B virus core and e antigens
INVENTOR(S): Sallberg, Matti
PATENT ASSIGNEE(S): Tripep AB, Swed.
SOURCE: PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001081421	A2	20011101	WO 2001-IB844	20010420
WO 2001081421	A3	20020404		

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6417324	B1	20020709	US 2000-556605	20000421

PRIORITY APPLN. INFO.: US 2000-556605 A 20000421

AB The present invention relates generally to the field of virol. More particularly, the invention relates to the discovery that peptides, which bind to the Hepatitis B virus (**HBV**) core and e antigens, can be used to **inhibit HBV** infection. Embodiments concern "binding partners," which include peptides, peptidomimetics, and chems. that resemble these mols. that interact with **HBV** core and e antigens, biol. complexes having **HBV** core and e antigens joined to said binding partners, methods of identifying such binding partners, pharmaceuticals having binding partners, and methods of treatments and prevention of **HBV** infectio

L17 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:458401 CAPLUS
DOCUMENT NUMBER: 138:100375
TITLE: Characterizing antiviral activity of adefovir dipivoxil in transgenic mice expressing hepatitis B virus
AUTHOR(S): Julander, Justin G.; Sidwell, Robert W.; Morrey, John D.
CORPORATE SOURCE: Animal, Dairy, and Veterinary Sciences Department, Institute of Antiviral Research, Utah State University, Logan, UT, 84322-4700, USA
SOURCE: Antiviral Research (2002), 55(1), 27-40
CODEN: ARSRDR; ISSN: 0166-3542
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Oral adefovir dipivoxil (ADV) reduced viral load in transgenic mice expressing hepatitis B virus (**HBV**). Liver **HBV** DNA was reduced to <0.1 pg of viral DNA per μg of total DNA (pg/μg) following oral ADV therapy at a dosage of 100 mg/kg/day twice daily for 10 days as compared to a mean of 3.0 pg/μg for the placebo control group. Oral ADV treatment also reduced serum **HBV** DNA to 3.5 log10 genomic equivalent (ge)/mL compared to 5.3 log10 ge/mL for the placebo control group. With once daily treatments, ADV antiviral activity reached near maximum viral reduction by day 10 in the liver and reached an endpoint of liver virus inhibition at 1.0 mg/kg/day. The min. ED was less than 0.1 mg/kg/day using inhibition of serum virus. Lamivudine (3TC) given orally at 500 mg/kg/day using the same treatment schedule marginally reduced the serum **HBV** DNA by 4-fold, but did not significantly reduce **HBV** liver DNA. Serum titer reduction was also identified in untreated or placebo-treated animals, which may have been caused by the stress of pre-treatment bleeding and multiple oral gavage treatments. This trauma/placebo-effect may have masked the extent of viral reduction in the serum in ADV- and 3TC-treated animals. Liver **HBV** RNA was not reduced by oral ADV treatments. The lack of RNA reduction was expected, because the **HBV** transgene is stably integrated into the chromosome and ADV **inhibits** polymerase activity after transcription of pregenomic RNA. ADV was identified to have potent anti-**HBV** activity in this **HBV** transgenic mouse model and could serve as a suitable pos. control for future drug discovery expts.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

DOCUMENT NUMBER: 137:348398
TITLE: Recombinant expression of human **HBV** reverse transcriptase (RT) for Phyllanthus amarus-derived antiviral **drug screening**
INVENTOR(S): Buchner, Johannes; Muschler, Paul; Halsbeck, Martin
PATENT ASSIGNEE(S): Phytrix A.-G., Germany
SOURCE: PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002088343	A2	20021107	WO 2002-EP4642	20020426
WO 2002088343	A3	20030424		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1390481	A2	20040225	EP 2002-740526	20020426
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: EP 2001-110073 A 20010427
WO 2002-EP4642 W 20020426

AB The present invention relates to a method of producing a functional cell-free hepatitis B virus (**HBV**) reverse transcriptase (RT) comprising the steps of expressing **HBV**-RT in E. coli cells employing a suitable expression plasmid, or in a cell-free transcription-translation system and if expression was carried out in E. coli cells, lysing said E. coli cells and purifying **HBV**-RT from E. coli lysate or from said transcription-translation system. The invention further relates to a method of screening for an inhibitor of **HBV**-RT activity comprising the steps of contacting the cell-free **HBV**-RT produced according to methods of the present invention and with a potential inhibitor and assaying whether said potential inhibitor **inhibits HBV**-RT activity. Furthermore, the invention provides for a method of producing a pharmaceutical composition comprising the step of formulating the inhibitor identified by the screening method of the invention into a pharmaceutical compn

L17 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:869219 CAPLUS
DOCUMENT NUMBER: 137:363028
TITLE: **Drug screening** assays and kits for discovery of anti-microbial and chemotherapeutics agents
INVENTOR(S): McCarthy, Lawrence; Kong, Lilly; Shao, Tang; Su, Xin
PATENT ASSIGNEE(S): Focus Technologies, Inc., USA
SOURCE: PCT Int. Appl., 94 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002090993	A2	20021114	WO 2001-US44783	20011127
WO 2002090993	A3	20040415		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003039957	A1	20030227	US 2001-996187	20011127

PRIORITY APPLN. INFO.:

US 2000-253150P	P	20001127
US 2001-304533P	P	20010709
US 2001-297686P	P	20010712
US 2001-996187	A2	20011127

AB Methods and compns. for detecting the phenotype of a bioactive mol. assays. More specifically, are provided methods and compns. are provided for determining the suitability of one ore more candidate compds. prior to or during the course of chemotherapy or anti-infective therapy, for their capacity to **inhibit** the bioactive mols. of micro-organisms, cancers and as an assay for expression in transgene therapy. Also provided are phenotypic assays for drug discovery. Claimed sequences were not present at the time of publication.

L17 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:368930 CAPLUS
DOCUMENT NUMBER: 136:374804
TITLE: Synthetic peptides that bind to the hepatitis B virus core and e antigens
INVENTOR(S): Sallberg, Matti
PATENT ASSIGNEE(S): Swed.
SOURCE: U.S. Pat. Appl. Publ., 56 pp., Cont.-in-part of U.S. Ser. No. 556,605.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002058247	A1	20020516	US 2001-839447	20010420
US 6417324	B1	20020709	US 2000-556605	20000421
US 2003235815	A1	20031225	US 2003-369060	20030214
US 2003225251	A1	20031204	US 2003-372735	20030221

PRIORITY APPLN. INFO.:

US 2000-556605	A2	20000421
WO 1995-SE468	W	19950427
US 1996-737085	A1	19961227
US 1999-246258	A1	19990208
US 2000-532106	A1	20000321
US 2000-664025	A2	20000919
US 2000-664945	A2	20000919
US 2001-839666	A1	20010419
US 2001-839447	B1	20010420
WO 2001-IB2327	A2	20010919
US 2002-153271	A2	20020521
US 2002-234579	A2	20020830

AB The present invention relates generally to the field of virol. More particularly, the invention relates to the discovery that peptides, which bind to the Hepatitis B virus (**HBV**) core and e antigens, can be used to **inhibit HBV** infection. Embodiments concern "binding partners", which include peptides, peptidomimetics, and chems. that resemble these mols. that interact with **HBV** core and e antigens, biol. complexes having **HBV** core and e antigens joined to said binding partners, methods of identifying such binding partners, pharmaceuticals having binding partners, and methods of treatments and prevention of **HBV** infectio

L17 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:398699 CAPLUS
DOCUMENT NUMBER: 131:165030
TITLE: Combinatorial screening and intracellular antiviral activity of hairpin ribozymes directed against hepatitis B virus
AUTHOR(S): Putlitz, Jasper zu; Yu, Qiao; Burke, John M.; Wands, Jack R.
CORPORATE SOURCE: Molecular Hepatology Laboratory, Massachusetts General Hospital Cancer Center, Harvard Medical School, Boston, MA, 02129, USA
SOURCE: Journal of Virology (1999), 73(7), 5381-5387
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A combinatorial screening method has been used to identify hairpin ribozymes that **inhibit** hepatitis B virus (**HBV**) replication in transfected human hepatocellular carcinoma (HCC) cells. A hairpin ribozyme library (5+105 variants) containing a randomized substrate-binding domain was used to identify accessible target sites within 3.3 kb of full-length in vitro-transcribed **HBV** pregenomic RNA. Forty potential target sites were found within the **HBV** pregenomic RNA, and 17 sites conserved in all four subtypes of **HBV** were chosen for intracellular inhibition expts. Polymerase II and III promoter expression constructs for corresponding hairpin ribozymes were generated and cotransfected into HCC cells together with a replication-competent dimer of **HBV** DNA. Four ribozymes inhibited **HBV** replication by 80, 69, 66, and 49%, resp., while catalytically inactive mutant forms of these ribozymes affected **HBV** replication by 36, 28, 0, and 0%. These findings indicate that the inhibitory effects on **HBV** replication were largely mediated by the catalytic activity of the ribozymes. In conclusion, the authors have identified catalytically active RNAs by combinatorial screening that mediate intracellular antiviral effects on **HBV**.
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT